the degree of preservation of useful vision afterward. The fact that some of the most serious eye injuries result from scleral rupture, after which one may be unable to observe extrusion of eye contents until after exploration of the globe and orbit, is not discussed.

Before surgery for penetrating eye injury, adequate examination is often not feasible until after the patient is anesthetized, prepped, and draped. Prolapse of eye contents is not unusual. Just because the surgeons in Libonati et al.'s report did not complain of extrusion after anesthetic induction and use of succinylcholine does not mean that there was none or that there was no additional loss of contents.

We are fortunate to have alternatives to use of succinylcholine for intubation. Given the potential for good visual outcome, we feel that the literature supports evidence for avoidance of use of succinylcholine in penetrating eye injuries.\(^7,8\)

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**Assay for Serum Sufentanil Level Is Not Sensitive**

**To the Editor:**—We agree with Weldon et al.\(^1\) that there are no assays that allow one to estimate accurately the elimination clearance of sufentanil after small standard doses. Unfortunately, their capillary gas chromatographic method, as it is presented,\(^1\) does not seem to change this situation and may actually lend confusion to what might otherwise have been a straightforward clinical report.\(^2\)

The most serious deficiency in the report of this new
assay is that the precision and accuracy of this method over the claimed range of linearity (0.03–5.0 ng sufentanil/ml) are not reported. Neither are the data necessary to validate quantitation of sufentanil metabolites presented. Data should be given for analyses of replicate sets of standard samples at concentrations that span the range of linearity of the methods to demonstrate their validity.

To evaluate the method, blood samples were collected from a patient for 24 h. Data are presented for samples obtained during the first 150 min, at which time the lower limit of detection was reached. A careful reploting of the data in the traditional log plasma concentration versus time manner reveals that the terminal elimination (beta) phase was just beginning at 150 min. Thus, even if this method were accurate and precise, it would not permit estimation of the elimination clearance of sufentanil except at doses three to four times those used in their study. The authors suggest that extracting larger plasma volumes might alleviate this problem but the "occasional interferences by substances of unknown origin or structure" seen in extracts of 2 ml plasma aliquots may be more severe when three to four times as much plasma is extracted. The interferences may be related to the splitless injection of large volumes (5–8 \( \mu l \)) onto a capillary column. Unfortunately, injection of a smaller volume would further limit sensitivity of the assay.

In the same issue, Wiggum et al.\(^2\) report plasma sufentanil concentrations in a chronic renal failure patient in whom anesthesia was induced with sufentanil (1.5 \( \mu g/kg \)) and thiopental (1.25 mg/kg). At 5, 9, and 13 h after the administration of sufentanil, plasma concentrations were 2.6, 1.2, and 0.6 ng/ml, respectively, suggesting to the authors that the elimination half-life was elevated beyond the 164 ± 70 (mean ± SD) min reported in normal patients.\(^3\) While this interpretation is questionable (the estimated half-life, 240 min, seems to be approximately one SD above the mean reported by Bovill et al.), it is more disturbing that they were unable to measure the plasma sufentanil concentration 4 h later. This is despite the fact that the predicted plasma concentration, 0.3 ng/ml, is more than one order of magnitude higher than the claimed lower limit of detection (0.02 ng/ml). What were the investigators measuring at 5, 9, and 13 h? Alternatively, why were they not able to detect sufentanil at 17 h? How do these discrepancies change their interpretation of the patient's apparent problems?

Variation in the metabolism of alfentanil due to the existence of polymorphic oxidation phenotypes has been suggested\(^4,5\) but has not been unequivocally demonstrated. Such should certainly not be invoked in these early stages of the study of sufentanil to explain possibly reduced clearance of this drug.\(^1,2\)

Finally, the authors\(^2\) do not present convincing evidence of narcotic-induced respiratory depression in this patient who complained of dyspnea (i.e., was awake) 10 min after arriving in the recovery room with a respiratory rate of 16 breaths/min. No further reference was given to any ventilatory measurements. In addition, the patient had decreased mentation and a Pa\(_{CO_2}\) of 64 mmHg, neither of which corresponded to the administration of 0.4 mg of naloxone. His mental status apparently improved with intubation and mechanical ventilation, a situation more consistent with primary respiratory embarrassment and subsequent CO\(_2\) narcosis than it is with primary sufentanil-induced narcosis and subsequent respiratory depression. Complaints of dyspnea, CO\(_2\) retention, and apparently decreased FRC (Pa\(_{CO_2}\) 96 mmHg on 100% \( O_2\) ) might be easily explained on the basis of a primary respiratory embarrassment resulting from acute abdominal distention by a large volume of peritoneal dialysis fluid instilled following placement of the peritoneal dialysis catheter.

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